Aedes aegypti in Tucson, Arizona

To the Editor: The highly domestic mosquito species *Aedes aegypti,* a tropical, nonnative vector of dengue and yellow fever, has been identified in several desert communities, including the city of Tucson and the border towns of Douglas, Naco, and Nogales (1). *Ae. aegypti* has now been found in the southern Arizona communities of Benson and Sahuarita Heights, which indicates that its distribution is probably expanding. Moreover, mosquito surveillance indicating that *Ae. aegypti* populations in Tucson are established throughout the city supports local concerns that the mosquito poses a public health risk in Arizona's second largest metropolitan area.

Since it was first detected in 1994 on Tucson's west side, the mosquito has been found in the central downtown and university districts, northern foothills, and the east and south sides (both in residential areas, including schools and parks, and in business districts). Between 1995 and 1997, almost 200 adult *Ae. aegypti* (female and male) were recovered in CDC CO_2 traps from 26 sites in Tucson by vector biologists from the Arizona Department of Health Services, Pima County, and the University of Arizona Veterinary Science Department.

These trapping events occurred between May and October of each year and were associated with either routine arbovirus surveillance or nonsystematic *Ae. aegypti* "spot-checks." Many of the sites were sampled more than once and 30% contained *Ae. aegypti* on multiple occasions. Catches from individual sites contained 1 to 40 adult mosquitoes (mode = 1).

A larval survey of the city has not yet been conducted because of limited staff and problems associated with oviposition trapping, an important part of *Ae. aegypti* larval surveillance (2). Initial attempts at ovitrapping by state, county (1), and later university personnel were not successful because the hay infusion water in ovitraps evaporated rapidly in Tucson's arid climate. However, the few larvae recovered from household containers suggest that Tucson's urban environment is providing a breeding habitat. Since the local climate requires full-time vigilance of ovitraps, this method of surveillance appears too labor intensive for the present.

In 1997, the University of Arizona Entomology Department initiated a mosquito survey of the city. This multiyear project is funded by the Entomology Department, the city of Tucson, and the Pima County Health Department. Neighborhood associations in Tucson were surveyed for their perceptions of the magnitude of the mosquito problem in their areas. On the basis of survey results, the city was divided into regions: north, east, south, west, and central. Four trapping stations were established in each region for a total of 20 sites spanning the metropolitan Tucson and outlying areas. The five regions were surveyed for mosquitoes through use of CO_2 traps approximately every 10 days starting July 1, 1997; traps were set in the late afternoon and collected in the late morning. Daytime CO_2 trappings were not effective.

The Entomology Department's 1997 surveillance data suggest that the central part of the city is the most heavily infested. Of 95 adult *Ae. aegypti* trapped, 49.5% were from the central region of Tucson, 18.9% from the west side, 17.9% from the east side, 10.5% from the north side, and 3.2% from the south side. The mosquito populations appeared to fluctuate with the weather, increasing in size after rainfall. Long-term trapping and future larval surveys should shed more light on this association.

Pima County and the University of Arizona Veterinary Science Department trapping activities in 1997 also produced evidence of *Ae. aegypti* in two communities near Tucson. Six adult mosquitoes were recovered in the town of Benson, 30 miles southeast of Tucson, and seven were trapped in Sahuarita Heights, 15 miles south of Tucson. The presence of the mosquitoes in these communities, as well as in Douglas, Naco, and Nogales, demonstrates that Arizona's smaller desert communities are also susceptible to *Ae. aegypti* infestations. The humidity emitted by home evaporative coolers may be crucial for the survival of tropical mosquitoes, such as *Ae. aegypti,* in Arizona's arid climate (N. Monteny, pers. comm.).

Genetic analysis of the *Ae. aegypti* collected from southeastern Arizona, Texas, and Mexico is under way at the University of Arizona Ecology and Evolutionary Biology Department to determine the structure, history, and origin(s) of the reemergent mosquito populations. Preliminary findings from mitochondrial DNA sequences suggest that *Ae. aegypti* in Arizona represent a single (panmictic) population, which indicates frequent local migration. More extensive sampling is necessary to confirm these results and determine a point of origin.

A community outreach program has been developed to inform the public about *Ae. aegypti* breeding and control in Tucson. Public involvement will be a key factor in the control of these urban breeders. Major emphasis will also be placed on programs for children and teachers as both groups can be instrumental in maintaining long-term interest in this problem. As these programs are developed, they can be expanded and amended to meet the needs of other infested communities in southern Arizona. A mosquito control abatement district is under consideration in a central part of Tucson. The primary purpose of this district would be to provide approximately 10,000 homeowners with information on controlling *Ae. aegypti* breeding on their property.

Just how long the *Ae. aegypti* infestation will last is difficult to assess. Records of the city's earlier infestation indicate the mosquito was present for at least a 15-year period (1931 to 1946) (1,3,4). Since their identification in early 1998 summer mosquito samples from Tucson, adult *Ae. aegypti* have been part of the city's local environment for at least 5 consecutive years (1994 to 1998). Their continued presence and the abundant breeding habitat provided by the expansion of Tucson's urban landscape suggest that *Ae. aegypti* could survive for an extended period.

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References

1. Engelthaler DE, Fink TM, Levy CE, Leslie MJ. The reemergence of *Aedes aegypti* in Arizona. Emerg Infect Dis 1997;3:241-2.
2. Reiter P, Amador MA, Colon N. Enhancement of the CDC ovitrap with hay infusions for daily monitoring of *Aedes aegypti* populations. J Am Mosq Control Assoc 1991;7:52-5
3. Murphy D. Collection records of some Arizona mosquitoes. Entomological News 1953;14:233-8.
4. Bequaert J. *Aedes aegypti*, the yellow fever mosquito, in Arizona. Bulletin of the Brooklyn Entomological Society 1946;41:157.

Can the Military Contribute to Global Surveillance and Control of Infectious Diseases?

To the Editor: Numerous networks—both formal (e.g., Ministries of Health and WHO Collaborating Centers and collaborating laboratories) and informal (e.g., nongovernmental and humanitarian organizations, the media, and electronic discussion groups)—contribute to WHO's network of networks for the global surveillance of infectious diseases (1).

A potential source of additional information on infectious diseases is the network of military health facilities and laboratories throughout the world. In addition to health facilities serving populations at high risk for infectious diseases, the military also has laboratories, often among the better-equipped, in developing countries. To evaluate the feasibility and potential usefulness of including military laboratories in the WHO global surveillance network, we conducted three surveys.

The first survey identified military laboratories willing to participate in global surveillance activities and obtained information about their infectious diseases reporting systems. Of the 107 countries surveyed, 76 replied. Among them, 53 (70%) reported having a central military laboratory that coordinates laboratory activities throughout the military, and 62 (82%) reported that military clinical facilities had a reporting system for infectious diseases.

The second survey quantified laboratory capabilities in the 53 laboratories identified in the first survey and obtained details about the 62 reporting systems. Among the 39 (74%) laboratories that replied, all can perform at least one of the following activities: isolating and identifying bacterial, viral, or parasitic agents. Twenty-nine (55%) have the capacity for specialized immunologic or molecular study. In addition, one of these laboratories has a biosafety level 4 facility, six have a biosafety level 3 facility, and 10 have a biosafety level 2 facility. Twenty-seven (51%) of the laboratories perform compulsory screening of new recruits for HIV, 17 (33%) for hepatitis B, 7 (13%) for hepatitis C, 39 (74%) for tuberculosis, 35 (67%) for syphilis, 18 (34%) for intestinal parasites, 13 (25%) for schistosomiasis, 12 (23%) for malaria, and 2 (4%) for Chagas disease.

Among the 54 reporting systems for which further information was obtained, clinical diagnoses (in some countries laboratory confirmed) are reported through the hierarchical chain, normally by mail or facsimile, but in two countries by electronic links. Almost all military reporting systems are parallel to civilian systems. Thirty-four (63%) of 54 systems feed into the civilian system, with a built-in mechanism to avoid duplicate reporting; 16 (30%) systems feeding into the civilian system have no such mechanism in place; and four have no link with the civilian system.

The third survey addressed vaccination policies. Among 52 countries that replied, 47 (90%) have a compulsory military vaccination schedule: 45 (87%) for tetanus, 30 (58%) for diphtheria, 23 (44%) for typhoid, 16 (31%) for bacillus Calmette-Guérin and polio, 12 (23%) for meningococcal meningitis, and 10 (19%) for measles, mumps, and rubella.

These surveys show that military populations are protected against many infectious diseases and that a wealth of information is obtained by military laboratories and health-care facilities on populations at high risk for infectious diseases. While most of the information collected from the health-care facilities is reported through civilian systems as well, incorporating the military network of laboratories into the WHO global surveillance network could ensure broader coverage.

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Reference

1. Heymann DL, Rodier GG. Global surveillance of Communicable Diseases. Emerg Infect Dis 1998;4:362-5.

Dual Infection with *Ehrlichia chaffeensis* and a Spotted Fever Group Rickettsia: A Case Report

To the Editor: In their article, Daniel J. Sexton et al. state, "Well- documented cases of simultaneous human infections with more than one tick-borne pathogen are rare" (1) and mention only two reports of such cases. However, another report should be mentioned because of its historical interest and the lessons it may teach.

In 1900 to 1905, in the Bitter Root Valley, a tick-borne disease emerged, which became known as Rocky Mountain spotted fever. Although Ricketts et al. later published a report (2), which identified the causative agent, in 1904 L.B. Chowning and W.M. Wilson published Studies on *Pyroplasma hominis* (3). They reported finding *Pyroplasma* (since changed to *Babesia)* in the blood of approximately 20 patients with spotted fever. They studied this organism in detail and even found the reservoir for it in the local rodent species. Wilson et al. thought that the organism was the causative agent of spotted fever. On the basis of their excellent plates and descriptions, it is clear that the organism they were describing was what we later came to know as *Babesia microti.*

The work of Wilson and Chowning was ignored and forgotten for many years. They had incorrectly concluded that spotted fever was caused by a parasite. For many years it was "well known" that *Babesia* infections became apparent in human patients only on removal or inactivation of the spleen. That persons with functional spleens were subject to infection with *B. microti* was finally established by the so-called Nantucket outbreak (4) and subsequent publications.

Therefore, Wilson and Chowning's work reports several cases of simultaneous infections of humans by two tickborne pathogens; i.e., patients had spotted fever and *B. microti* in the blood. More poignant was that an "emerging" disease of humans was missed and not discovered again for some 70 years.

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References

1. Sexton DJ, Corey GR, Carpenter C, Kong LQ, Gandhi T, Breitschwerdt E, et al. Dual infection with *Ehrlichia chaffeensis* and a spotted fever group rickettsia: a case report. Emerg Infect Dis 1998;4:311-6.
2. Ricketts HT. Some aspects of Rocky Mountain spotted fever. Rev Infect Dis 1909;1227-40.
3. Wilson LB, Chowning WM. Studies on *Pyroplasma hominis.* Rev Infect Dis 1904;1:31-57.
4. Ruebush TK, Juranek DD, Chisholm ES, Snow PC, Healy GR, Sulzer AJ. Human babesiosis on Nantucket Island. N Engl J Med 1977;297:825-87.

Dual Infection with *Ehrlichia chaffeensis* and a Spotted Fever Group Rickettsia: A Case Report—Reply to Dr. Sulzer

To the Editor: Several investigators have suggested that some of Wilson and Chowning's patients may have had coinfection with *Babesia* and *Rickettsia rickettsii* (1-4). Furthermore, the organisms that Wilson and Chowning observed in red cells of 20% of the local Columbian ground squirrels are consistent with later reports of various species of *Babesia* in the erythrocytes of other species of squirrels (4). However, most rickettsiologists who have commented on Wilson and Chowning's paper have concluded that intraerythrocytic organisms observed in blood samples did not contribute substantially to the illnesses of the 23 patients described. Although Stiles, Wenyon, and Brumpt concluded that the organisms in human blood samples observed by Wilson and Chowning were artifacts or malarial parasites (5-7), contemporary experts who have reviewed the colored plates that accompanied Wilson and Chowning's 1904 paper believe that there is "little" or "no doubt" that Wilson and Chowning actually described organisms of the genus *Babesia* (1,2,8).

In a commentary that followed the republication of Wilson and Chowning's landmark paper in 1979 (9), Richard Ormsbee reviewed the sequence of events that followed the publication of Wilson and Chowning's report in 1904 (10). After more than 200 hours of careful microscopy, C.W. Stiles could find no evidence of *Pyroplasma* in the blood of 12 patients with Rocky Mountain spotted fever (RMSF). He refuted Wilson and Chowning's findings (5) and challenged Chowning, who was also in the Bitter Root Valley, to demonstrate the presence of organisms in the blood of a person with a typical case of RMSF. Chowning was unable to find *Pyroplasma* in blood smears from these patients (10). Ricketts did not arrive in the Bitter Root Valley to begin his studies of RMSF until 1906 (11); thus he could not have published his classic paper on the etiology of RMSF in volume 1 of the Journal of Infectious Diseases.

To our knowledge, ecologic studies done in the Bitter Root Valley have not demonstrated endemic foci of babesial infection. A serologic survey of 246 Bitter Root Valley residents in 1978 showed no antibabesial antibodies (12). Although it is possible that 4 of the 23 patients with RMSF described by Wilson and Chowning had incidental preexisting latent babesial infection, the clinical and autopsy data they presented suggest that the patients had typical *R. rickettsii* infection. There is no proof that any of the patients described by Wilson and Chowning had simultaneous acute babesial and rickettsial infection, and we agree with Ormsbee that the significance of the "*Pyroplasma hominis*" described in the blood smears of several of Wilson and Chowning's patients is "... a mystery that persists to this day" (10).

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References

1. Gorenflot A, Mourbri K, Precigout E, Carcy B. Human babesiosis. Ann Trop Med Paristol 1998;92:489-501.
2. Hoare CA. Comparative aspects of human babesiosis. Trans R Soc Trop Med Hyg 1980;74:143-8.
3. Ruebush TK II, Cassaday PB, Marsh HJ, Lisker SA, Voorhers D, Mahoney EB, et al. Human babesiosis on Nantucket Island. Clinical features. Ann Intern Med 1977;86:6-9.
4. Pinsky RL, Lutwick LI. Spotted fever and babesiosis. Rev Infect Dis 1979:1:895.
5. Stiles CW. A zoological investigation into the cause, transmission, and source of Rocky Mountain "spotted fever." Hygienic Laboratory Bulletin . Washington: Government Printing Office; 1905. Publication No 20:7-119.
6. Wenyon CM. Protozoology. Vol 2. London: W. Wood; 1926.
7. Brumpt E. Precis de parasitologie. 5th ed. Paris: Masson et Cie; 1936. p. 497.
8. Pruthi RK, Marshall WF, Wiltsie JC, Persing DH. Human babesiosis. Mayo Clin Proc 1995:70:853-62.
9. Wilson LB, Chowning WM. Studies in Pyroplasmosis hominis ('spotted fever" or "tick fever of the Rocky Mountains). Rev Infect Dis 1979;1:540-58.
10. Ormsbee RA. Studies in Pyroplasmosis hominis ("spotted fever" or "tick fever" of the Rocky Mountains) by Louis B. Wilson and William A. Chowning. Rev Infect Dis 1979;1:559-62.
11. Harden VA. Rocky Mountain spotted fever research and the development of the insect vector theory, 1900-1930. Bull Hist Med 1985;59:449-66.
12. Chisholm ES, Ruebush TK II, Sulzer AJ, Healy GR. *Babesia microti* infection in man: evaluation of an indirect immunofluorescent antibody test. Am J Trop Med Hyg 1978:27:14-9.

Reemergence of *Plasmodium vivax* Malaria in the Republic of Korea

To the Editor: In "Reemergence of *Plasmodium vivax* malaria in the Republic of Korea" (1), the term eradication was, in my judgment, inappropriately used. In 1981, Yekutiel proposed that eradication is "The purposeful reduction of specific disease prevalence to the point of continued absence of transmission within a specified area by means of a time limited campaign" (2). In 1984, Hinman proposed an important addition that eradication must have followed a "deliberate effort" (3). At the Dahlem Workshop in 1997 (4), a more comprehensive definition was proposed. This definition states that eradication is "Permanent reduction to zero of the worldwide incidence of infection caused by a specific agent as a result of deliberate efforts; intervention measures are no longer needed" (4). At the same conference, two other terms were also defined. Elimination of disease: "Reduction to zero of the incidence of a specified disease in a defined geographic area as a result of deliberate efforts; continued intervention measures are required." Elimination of infection: "Reduction to zero of the incidence of infection caused by a specific agent in a defined geographic area as a result of deliberate efforts; continued measures to prevent reestablishment of transmission are required."

These definitions promote unanimity in using the term eradication and avoid misconceptions over accomplishments.

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References

1. Feighner BH, Pak SI, Novakoski WL, Kelsey LL, Strickman D. Reemergence of *Plasmodium vivax* malaria in the Republic of Korea. Emerg Infect Dis 1998;2:295-7.
2. Yekutiel P. Lessons from the big eradication campaigns. World Health Forum 1981;2:465-81.
3. Himnan AR. Prospects for disease eradication or elimination. New York State General Medicine 1984;84:502-6.
4. Ottesen EA, Dowdle WR, Fenner F, Habermehl KO, John TJ, Koch MA et al. In: Eradication of infectious diseases. Dowdle WR, Hopkins DR, editors. New York: John Wiley and Sons; 1997. p. 48.

Paratyphoid Fever

To the Editor: The letter on paratyphoid fever by Kapil et al. (1) stated that an outbreak of enteric fever due to *Salmonella paratyphi* A has never been reported. A large (227 cases) outbreak of enteric fever secondary to *S. paratyphi* A occurred in the Arabian Gulf nation of Bahrain in 1987. The clinical and epidemiologic details of the outbreak were reported in a local medical society journal (2). Like the outbreak described by Kapil et al., the Bahraini outbreak was associated with sewage leaking into the water supply.

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References

1. Kapil A, Sood S, Reddaiah VP, Das B, Seth P. Parayphoid fever due to *Salmonella enterica* serotype paratyphi A. [Lett]. Emerg Infect Dis 1997;3:407.
2. Al-Madani R. Paratyphoid Outbreak in children. Journal of the Bahrain Medical Society 1989;1:60-3.

Hospitalizations After the Persian Gulf War

To the Editor: Knoke et al., Naval Health Research Center, San Diego, California, published two articles on military hospitalizations in Persian Gulf War veterans, the most recent in Emerging Infectious Diseases (1,2).

Although the titles of both articles indicated general hospitalizations, Knoke et al. studied just military hospitalizations among selected, mostly healthy, active-duty Persian Gulf War veterans enlisted as of 1994. They compared military hospitalizations of active-duty Gulf War veterans (cases) with military hospitalizations of active-duty era veterans not in the Persian Gulf between 1990 and 1991 (controls). "Healthy warrior" effects would have predicted low military hospitalization rates for both cases and control populations (3), but both were high.

The studies were "restricted to active-duty personnel" hospitalized in military facilities because active-duty personnel were "rarely hospitalized outside of DoD facilities" (1). However, of 150 surgical procedures, mostly intestinal and skin biopsies, performed on 85 sick active-duty and reservist Persian Gulf veterans from Pennsylvania between 1991 and 1995, more than one third, 58 (39%), were performed in private facilities (4). Most of the federal procedures were done in Veterans Administration (VA), not military, hospitals. Many active-duty, sick Persian Gulf veterans in Pennsylvania, Texas, and California deliberately avoided military, and some VA, hospitals between 1991 and 1997 because of concerns about competence, convenience, confidentiality, and career opportunities during this era of downsizing and closing of military bases (3,5).

In addition, Knoke et al. excluded at least five groups of sick veterans from their limited case studies: 1) those treated in VA and private hospitals, 2) those from the Reserves and National Guard, 3) those who retired early largely because of illness, 4) those who consented to long military hospitalizations within the DoD Comprehensive Clinical Evaluation Program (CCEP) for Gulf War Veterans, and 5) those who had obstetric complications after returning home from the Gulf War. Thus, many sick veterans were excluded from the case studies.

If we hypothesize that one or more new infectious agents like *Leishmania tropica, Brucella* species, *Bacillus anthracis, Mycoplasma fermentens (incognitus), Coxiella burnetti,* or obscure fungi or molds might be involved, comprehensive research studies in the future would do better to include all workers from the Arabian desert, reservists as well as active-duty personnel.

Few Gulf veterans with Gulf-related illnesses were welcomed by military hospitals and about half of 452 Persian Gulf veterans surveyed by the U.S. General Accounting Office sought health care outside the VA for health problems they believed were related to service in the Persian Gulf (5). An alternative interpretation of Knoke's hospitalization study might be that admitting officers in military facilities prevented sick Persian Gulf War veterans from obtaining medical care within their facilities.

Not only were the case populations studied unusual; recent workers and travelers to the Middle East were not excluded from the control population. "Nondeployed" controls included recently deployed Persian Gulf military personnel as long as nondeployed personnel worked in the Gulf after 1991. Some of those late-deployed Persian Gulf workers also fell ill with the same illnesses as veterans deployed between August 1990 and 1991. Illnesses from late Persian Gulf deployments might explain excess hospitalizations seen in nondeployed controls. All late-deployed personnel from the Middle East should also have been excluded from the nondeployed control population.

Finally, medical ICD-9 diagnoses, while interesting, were incomplete and nonspecific. Medical diagnoses common to Gulf veterans should have been listed in addition to unexplained illnesses. Knoke's condensed diagnostic list, like patient charts we have seen from DoD hospitalizations, may have failed to capture common clinical and laboratory abnormalities seen in many sick Gulf veterans, including (but are not limited to) ulcerative colitis, Crohn colitis, inflammatory bowel disease, intestinal bleeding due to inflammatory colonic polyps, skin acne, nodules, plaques, psoriasiform skin rashes, nose ulcers, nose bleeds, leukocytosis, neutropenia, elevated alanine transaminase (SGPT/ALT) liver enzymes, hepatosplenomegaly, thrombocytopenia, nephrolithiasis (kidney stones), and fevers of unknown origin (4,6). In addition, more than one unexplained illness category should have been tabulated per patient, because "Gulf War Syndrome" is a multisystem illness (4,6-9).

More research is needed on hospitalizations in addition to deaths and new diseases found in Persian Gulf War veterans (3). Civilian scientists and physicians must collaborate closely with other diverse federal and nonprofit organizations to study Gulf War illnesses objectively (5,9). The health problems seen in Gulf War veterans may be part of a new complex of emerging desert-associated illnesses (9-14).

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References

1. Gray GC, Coate BD, Anderson CM, Kang HK, Berg SW, Wignall RS, et al. The postwar hospitalization experience of U.S. Persian Gulf War veterans compared with other veterans of the same era. N Engl J Med 1996;335:1505-13.

2. Knoke JD, Gray GC. Hospitalizations for unexplained illnesses among U.S. veterans of the Persian Gulf War. Emerg Infect Dis 1998;4:211-19.
3. Haley RW. Bias from the "healthy warrior effect" and unequal follow-up in three government studies of health effects of the Gulf War. Am J Epidemiol 1998;148:315-23.
4. Murray-Leisure KA, Sees J, Zangwill B, Suguitan E, Legaspi C, Bagheri S, Mucha P, Brinser E, Kimber R, Kurban R, Greene W. Mucocutaneous-intestinal-rheumatic Desert Syndrome (MIRDS): Incubation & histopathology findings. Abstract K 48, American Society for Microbiology, ICAAC, Sept. 18, 1995.
5. U.S. General Accounting Office. Gulf War Illnesses: Improved Monitoring of Clinical Progress and Reexamination of Research Emphasis are Needed. GAO/NSIAD-97-163. June 1997.
6. Sostek MB, Jackson S, Linevsky JK, Schimmel EM, Fincke BG. High prevalence of chronic gastrointestinal syndromes in a National Guard unit of Persian Gulf Veterans. Am J Gastroenterol 1996;91:2494-7.
7. Centers for Disease Control. Unexplained illness among Persian Gulf War veterans in an Air National Guard unit: Preliminary report, Aug 1990-March 1995. Morb Mortal Week Rep 1995;44:443-7
8. Fukada K, Nisenbaum R, Stewart G, Thompson WW, Robin L, Washko R, et al. Chronic multisystem illnesses affecting Air Force veterans of the Gulf War. JAMA 1998;280:981-8.
9. Nicolson G, Nicolson N. Gulf War illnesses. Medicine, Conflict, and Survival 1998;14:156-65.
10. Magill AJ, Grogl M, Gasser RA, Wellington S, Oster CN. Viscerotropic leishmaniasis caused by *Leishmania tropica* in soldiers returning from Operation Desert Storm. N Engl J Med 1993;328:1383-7.
11. Magill AJ, Grogl M, Johnson SC, Gasser RA. Visceral infection due to *Leishmania tropica* in a veteran of Operation Desert Storm who presented two years after leaving Saudi Arabia. Clin Infect Dis 1994;19:805-6.
12. Ferrante MA, Dolan MJ. Q Fever meningoencephalitis in a soldier returning from the Persian Gulf War. Clin Infect Dis 1993;16:489-96.
13. Seaman J, Mercer AJ, Sondorp E. Epidemic of visceral Leishmaniasis in western upper Nile, southern Sudan: Course and impact from 1984 to 1994. Int J Epidemiol 1996;25:862-71.
14. Zilinskas RA. Iraq's biological weapons. JAMA 1997;278:418-24.

Hospitalizations after the Gulf War—Reply to K.M. Leisure et al.

To the Editor: We studied all active-duty Persian Gulf War–era veterans who remained on active duty at the conclusion of deployment (July 31, 1991), not as Leisure et al. stated in their letter "selected, mostly healthy, active-duty Persian Gulf War veterans enlisted as of 1994."

Our study was restricted to hospitalizations of active-duty service members because these were the only service members whose records were available on computerized files. No one was excluded from the defined target population. However, there are "sick Gulf War veterans" and healthy Gulf War veterans not in the target population. The difficulty is in studying either a random sample or the entire population of Gulf War veterans. The only published study we know of the entire population is the mortality report of Kang and Bullman (1).

The suggestion that we should have excluded from the control group service members who had ever been in the Gulf War area would have been appropriate for a report of exposure to the Persian Gulf region; ours was a report of exposure to the Persian Gulf War. That we should have studied a different collection of ICD-9 diagnoses also suggests a different report.

While our study may have limitations, we have not seen objective data that support the anecdotal observations of Leisure et al.

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Reference

1. Kang HK, Bullman TA. Mortality among U.S. veterans of the Persian Gulf War. N Engl J Med 1996;335:1498-504.